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Influenzal Meningitis: A Survey of Cases in a Small Hospital*

F. Clyde Bowers, M.D., Mendham, N. J.

In view of the many excellent reports in the medical literature in recent years on influenzal meningitis, it is with humility that this present effort is made. It was felt, however, that with the apparent increase in the number of cases being recognized today and further, that with the gratifying results obtained by the early institution of present day treatment, it might not be entirely amiss to survey and report the cases seen in a small institution in the past 9 years (1940–1949).

Etiology

Influenzal meningitis is an acute purulent meningitis due to the bacillus influenzae first described by Pfeiffer in 1892. No relationship exists between meningitis due to the B. influenzae and the so-called epidemic influenza which is due to a filterable virus.

The causative organism of the disease under discussion is a small, Gram negative, non-motile, hemophilic bacillus whose growth requirements are so fastidious as to make its isolation and recognition difficult. Pittmann in 1931 showed that the majority of Hemophilus influenza strains obtained from meningitis cases belong to a special group of this organism characterized by possessing a capsule consisting of carbohydrates. Six different types, A to F, have been differentiated and most of the meningitis strains belong to type B. However, Gordon et al[1] report 2 cases due to non-capsulated strains and not belonging to any of the Pittmann types. Fortunately, these strains have a lower virulence than the capsulated strains even though they are capable of causing meningitis in man. Sporadic cases due to capsulated types other than type B have been reported.[2,3]

Forty-six cases are herein presented, the diagnoses being proven by spinal fluid culture in all cases save one (no. 21). The responsible organism was Hemophilus influenza, type B in all cases except two (nos. 42 and 43) in which instances it was type A. It was believed that in seven cases (nos. 8, 13, 22, 23, 25, 27, 32) there was a concurrent infection with the meningococcus. Many may rightfully take issue with this finding since it is well known that the influenza bacillus stains well on its ends and sometimes is mistakenly identified as a coccus. Nevertheless, this finding is mentioned here because in most instances the first organism reported from the laboratory was the meningococcus. Sometimes it was not until several days later (cases 8, 13, 19) that the influenzal organism was identified. It becomes quite apparent, therefore, that in every case where prompt response to treatment is not obtained, especially in a reported meningococcus meningitis, one should resort to a diligent search for another organism, namely the H influenza.

*From the medical services of the Morristown Memorial Hospital, Morristown, N. J.

SPINAL FLUID

Number	*Age*	*Race Sex*	*Initial Symptoms*	*Hospital Stay*	*Cells*	*Protein*	*Sugar*	*Culture*	*Blood Culture*	*Treatment*	*Result and Comments*
1 G. A.	2	W M	Headache Vomiting 1 day	9 hours Jan. 1940	3900 polys.	xxxx	Neg.	Pos. B		Sulfapyridine	Died
2 F. M.	2½	W M	U. R. I. Vomiting 1 day	36 hours June 1940	22,000 polys.	xxxx	Neg.	Pos. B	Neg.	Sulfapyridine	Died
3 J. R.	1½	W F	Head injury 2 days later vomiting and drowsy	19 days Nov. 1941	13,000 polys.	250x	Neg.	Pos. B 19 days	Pos.	Sulfapyridine Sulfadiazine	Died
4 W. S.	5	W M	U. R. I. 3 days	60 days Aug. 1942	3240 polys.	Trace	Trace	Pos. B 8 days	Neg.	Sulfapyridine Sulfadiazine Serum-75 mgm. Blood trans	Died. Spinal fluid neg. and temp. normal on 8th day. Rx stopped on 16th day. On 19th day had recrudescence of fever and symptoms with positive culture
5 J. C.	1½	W F	U. R. I. 2 days	38 days Oct. 1943	14,000 polys.	164	Neg.	Pos. B 5 days		Sulfadiazine Sulfameradiazine	Died. Cell count dropped to 20 and culture neg. on 14th day but had recrudescence in spite of therapy
6 B. W.	3	W M	U. R. I. 3 days	26 days Dec. 1943	18,750 polys.	143	Neg.	Pos. B 1 day		Sulfadiazine Serum-125 mgm.	Recovered. Spinal fluid neg. after 1st day. Made uneventful recovery but 6 days after discharge was struck on head and re-admitted. Recovered without further treatment (2nd adm. 32 days)
7 L. C.	2	W F	Vomiting and listlessness 2 days	26 hours May 1944	960 polys.	102	Neg.	Pos. B 2 days		Sulfadiazine	Died

8 B. B.	4	W M	Chicken Pox—4 days before fever and stiff neck for 12 hours	38 days Oct. 1944	19,000 polys.	250x	Neg.	Pos. B 1 day and Meningococci		Sulfadiazine Serum-100 mgm. Penicillin Blood transfusions	Recovered 17 days after admission spinal fluid pos. for B. influenza for the first time
9 M. N.	16 mos.	W F	Whooping Cough onset 3 weeks previously. Day of admission was listless and had stiff neck	63 days Jan. 1945	2080 polys.		26	Pos. B 2 days		Sulfadiazine Serum-150 mgm.	Recovered. On 24th day abscess of left thigh. On 44th day abscess of right thigh. On 60th day bilat. Otitis Media
10 J. K.	1	W M	Vomiting Stiff neck 2 days	32 days Jan. 1945	8579 polys.	70	Below 20	Pos. B 1 day		Sulfadiazine Serum-70 mgm.	Recovered. Was given intrathecal serum and went into anaphylactic shock but recovered
11 Z. F.	3½	W M	Rhinitis 3 days	47 days Feb. 1945	1630 polys.	169	Below 20	Pos. B 2 days		Sulfadiazine Serum-121 mg n. Penicillin	Recovered. On 26th day of illness developed Chicken Pox
12 P. R.	4	W F	U. R. I. 5 days	20 days Oct. 1945	580 polys.	30	50	Pos. B 2 days	Neg.	Sulfadiazine Serum-125 mgm. Penicillin	Recovered. Temp. 106.6 on 2nd day—subsided by lysis
13 T. M.	46	W M	Headache and stiff neck 5 days	25 days Dec. 1945	15,250 polys.	250	Below 20	Pos. B 2 days and Meningococci 7 days		Sulfadiazine Serum-225 mgm. Penicillin	Recovered. Meningococci found in spinal fluid on admission. Influenzal bacilli found on fifth day

SPINAL FLUID

Number	*Age*	*Race Sex*	*Initial Symptoms*	*Hospital Stay*	*Cells*	*Protein*	*Sugar*	*Culture*	*Blood Culture*	*Treatment*	*Result and Comments*
14 R. B.	2	W M	Rhinitis 4 days	44 days Jan. 1946	4570 polys.	54	Too low to read	Pos. B 1 day		Sulfadiazine Serum-150 mgm. Penicillin Blood transfusions	Recovered. Convulsions followed by right hemiparesis which remained at discharge
15 B. W.	5	W F	Headache Vomiting 1 day	20 days Feb. 1946	5660 polys.	236	30	Pos. B 1 day		Sulfadiazine Serum-150 mgm. Penicillin	Recovered. Prompt response. Culture of spinal fluid not positive until 3rd day
16 Z. W.	28	B F	Headache Vomiting 1 day	8 days Mar. 1946	14,250 polys.	Over 273	2	Pos. B 5 days		Sulfadiazine Serum-400 mgm. Penicillin Streptomycin Blood transfusions	Died. Never showed signs of improvement. Guinea pig inoculation neg.
17 B. R.	9	W F	Headache Drowsy 1 day	21 days Mar. 1946	Bloody tap		65	Pos. B 1 day		Sulfadiazine Serum-75 mgm. Penicillin	Recovered. Prompt response
18 F. D.	29	W M	Headache 4 days	12 days May 1946	5000 polys.	69	95	Pos. B 1 day		Sulfadiazine Serum-100 mg. Penicillin	Recovered. Prompt response Fever 1 day

19 B. G	8	W F	Headache Vomiting 1 day	22 days Oct. 1946	65,000 polys.		101	Pos. B 1 day		Sulfadiazine Serum-175 mgm. Penicillin Blood transfusions	Recovered. Petechial hemorrhages in eyes. Causative organism not found until 6th day—then prompt response to serum
20 T G.	8	W M	Headache Fever 3 days	24 days Oct. 1946	650 polys.	69	93	Pos. B 2 days	Neg.	Sulfadiazine Serum-125 mgm. Penicillin	Recovered. Prompt response. Exposed to case of Influenzal Meningitis
21 H. B.	5	W M	Fever Vomiting 2 days	20 days Nov. 1946	160 polys.	54	105	No growth		Sulfadiazine Serum-25 mgm. Penicillin	Recovered. Diagnosis of Influenzal Meningitis questionable
22 R. E.	7	W M	General malaise 5 days	21 days Nov. 1946	7640 polys.	45	113	Pos. B 1 day and Meningococci		Sulfadiazine Serum-50 mgm. Penicillin Streptomycin	Recovered. Prompt response.
23 C. D.	19	W F	U. R. I.—2 weeks Vomiting 1 day Stupor	25 days Dec. 1946	7610 polys.	Too high to read	97	Pos. B 2 days and Meningococci	Pos. Infl.	Sulfadiazine Serum-150 mgm. Penicillin	Recovered. Acutely ill on admission. T. 106° 2 mos. post-partum and a nursing mother. Was stuporous for 48 hrs.
24 D. M.	9	W M	Abdominal pain and vomiting—4 days Headache—2 days	23 days Mar. 1947	350 polys.	132	27	Pos. B 23 days	Pos.	Sulfadiazine Serum-875 mgm. Streptomycin Blood trans.	Died. Acutely ill, delirious and confused with spiking temp. all during hospitalization. P. M. Acute Basilar Meningitis

SPINAL FLUID

Number	*Age*	*Race Sex*	*Initial Symptoms*	*Hospital Stay*	*Cells*	*Protein*	*Sugar*	*Culture*	*Blood Culture*	*Treatment*	*Result and Comments*
25 D. M.	8 mos.	W F	Drowsiness Temp. 8 hrs.	30 days Mar. 1947	3080 polys.	220	30	Pos. B 22 days and Meningococci		Sulfadiazine Serum-100 mgm. Penicillin Streptomycin	Recovered. Spinal fluid positive for 22 days. Temp. fluctuated until serum was given on 8th day and then dropped by lysis
26 A. M.	46	W F	Headache—3 weeks Stiff Neck—1 day	18 days Mar. 1947	88 polys.	118	29	Pos. B 18 days	Neg.	Sulfadiazine Serum-600 mgm. Streptomycin	Died. Acutely ill and never showed signs of response to therapy P. M. showed diffuse meningitis due to type B influenza
27 E. C.	14	W M	Headache—5 days	55 days May 1947	2390 polys.	250	Too low to read	Pos. B 55 days and Meningococci	Pos. Infl.	Sulfadiazine Serum-725 mgm. Penicillin Streptomycin Blood trans.	Died. Acutely ill. T. 106°. Convulsions. Spinal fluid pos. for 19 days, neg. for 17 days and pos. again. Was neg. for 7 days when Rx discontinued. O.K. for 10 days and then recrudescence. P. M. purulent meningitis
28 G. A.	29	W M	Headache—2 days	59 days July 1947	398 polys.	57	76	Pos. B 5 days		Sulfadiazine Serum-850 mgm. Streptomycin Blood trans.	Recovered. Acutely ill—fed by vein 3 weeks. Spinal fluid pos. for 39 days
29 D. D.	5	W F	Headache 3 days Otitis Media 10 days	6 hours July 1947	455 lymphs	240	54	Pos. B Sp. Fl. Ear Mastoid		Sulfadiazine Penicillin	Died. Had repeated convulsions following spinal tap. P. M. revealed acute purulent meningitis, mastoiditis, otitis media

30 A. K.	27	W F	U. R. I.— 4 days	17 days Aug. 1947	280 lymphs	270	63	Pos. B 7 days Sp. Fl. throat		Strepto- mycin	Recovered. Not acutely ill
31 R. R.	25	W F	Headache Stiff Neck 7 days	9 days Sept. 1947	480 lymphs	169	71	Pos. B 8 days	Neg.	Sulfadia- zine Serum-100 mgm. Strepto- mycin Blood trans.	Recovered. Transferred to another hospital on 9th day
32 E. G.	33	W F	U. R. I.— 2 weeks Otitis Media 24 hours	22 days Oct. 1947	6940 polys.	Too high to read	Too low to read	Pos. B 7 days Sp. Fl. Ear Men- ingo- cocci	Pos.	Sulfadia- zine Serum-500 mgm. Penicillin Strepto- mycin Blood Trans.	Recovered. Delirium 1 day 30 hrs. after admission was delivered of a full term normal living baby Sequela left facial paralysis
33 L. H.	7 mos.	W M	U. R. I. 24 hours	40 days Nov. 1947	1669 polys.	280	12	Pos. B 4 days		Sulfadia- zine Serum-200 mgm. Strepto- mycin Blood trans.	Recovered. Acutely ill Convulsions for 5 days with opisthotonus
34 J. P.	2 mos.	W M	Fever–1 day Convulsions– 1 day	30 days April 1948	40,000 polys.	165	120	Pos. B 3 days		Sulfadia- zine Serum-60 mgm. Penicillin Strepto- mycin Bl. Trans.	Recovered. Acutely ill. Convulsions for 2 days. Was moribund on admission

SPINAL FLUID

Number	*Age*	*Race Sex*	*Initial Symptoms*	*Hospital Stay*	*Cells*	*Protein*	*Sugar*	*Culture*	*Blood Culture*	*Treatment*	*Result and Comments*
35 W. S.	8	W M	Headache Nausea Vomiting 3 days	16 days April 1948	1120 lymphs	78	80	Pos. B 1 day		Sulfadiazine Serum-50 mgm. Penicillin Streptomycin	Recovered. Mumps 10 days before admission
36 R. M.	4	W M	Headache 1 day	14 days April 1948	650 polys.	22	74	Pos. B 1 day		Sulfadiazine Penicillin Streptomycin	Recovered. No intrathecal medication. Fever for only 24 hours
37 J. H.	18 mos.	W M	Fever, Stupor 3 days	17 days Sept. 1948	7050 lymphs	55	62	Pos. B 1 day		Sulfadiazine Serum-40 mgm. Streptomycin	Recovered. Uneventful—only slight fever for 3 days
38 J. M.	20 mos.	W F	Fever, Stiff Neck 1 day	14 days Dec. 1948	8200 polys.	66	100	Pos. B 7 days		Sulfadiazine Serum-25 mgm. Penicillin Streptomycin	Recovered. High temperature for 2 days—then uneventful
39 E. W.	5 mos.	C F	U. R. I. Fever 5 days	24 days Dec. 1948	7900 polys.	188	40	Pos. B 2 days	Neg.	Sulfadiazine Serum-150 mg. Penicillin Streptomycin	Recovered. Convulsion—1 day. Temperature persisted for ten days
40 J. C.	56	W F	U. R. I. 1 week	10 days Dec. 1948	1570 polys	536	20	Pos. B 9 days	Neg.	Sulfadiazine Serum-200 mgm. Penicillin Streptomycin Bl. Trans.	Died. Proper Rx delayed too long. Had T. 104° for 2 days before admission—also bronchopneumonia

41 P. H.	14	W M	Rhinitis Convulsion 1 day	33 days Feb. 1949	180 lymph	76	81	Pos. B 10 days	Neg.	Sulfadiazine Serum-400 mgm. Streptomycin Bl. Trans.	Recovered. Acutely ill—irrational for 5 days and convulsions. Spinal fluid positive for 10 days—Vestibular nerve involvement
42 H. L.	52	W F	U. R. I. 1 week	33 days Feb. 1949	345 polys.	280	16	Pos. A 7 days		Sulfadiazine Penicillin Streptomycin	Recovered. Irrational for 2 days
43 B. S.	4 mos.	W F	Fever Convulsions 1 day	49 days Feb. 1949	2580 polys.	110	57	Pos. A 1 mo.		Sulfadiazine Serum-75 mgm. Penicillin Streptomycin	Recovered. Culture remained positive for 1 month. Lethargy for 5 days
44 T. B.	18 mos.	W M	U. R. I. 1 week	22 days Mar. 1949	3600 polys	48	20	Pos. B 7 days		Sulfadiazine Serum-125 mgm. Streptomycin	Recovered. Also had acute Otitis Media
45 H. W.	49	W F	Pain in neck 5 days	18 days May 1949	Bloody tap	48	45	Pos. B 3 days		Sulfadiazine Streptomycin	Died. No Streptomycin intrathecally. Was almost clinically well when suddenly died of cerebral hemorrhage
46 I. W.	60	W M	U. R. I. 7 days	39 days May 1949	120 lymphs	10	66	Pos. B 10 days		Sulfadiazine Serum-250 mgms. Streptomycin Bl. Trans. Aureomycin Chloromycetin	Recovered. Irregular, fluctuating temp. for 27 days. Difficult to evaluate effectiveness of one medication

Frequency

As recent as 1931 Todd and Sanford noted that the influenzal bacillus was rarely found as the causative organism in meningitis. Two years later Griffith and Mitchell mentioned it as a more frequent offender in infants and children but it held the unenviable position of being almost a 100% killer. At the present time it ranks first as the etiological factor in purulent meningitis in many clinics; for example, in 1948 Martinez[4] revealed that in 340 cases of meningitis, the causative organism was found to be H influenza in 212.

It has always been felt that influenzal meningitis was an affliction of children especially under five years of age, the percentages running as high as 85%.[2,3–6] One explanation for this was that children over five, and especially adults, had a certain amount of immunity to the influenzal bacillus and possessed enough antibodies to avoid infection. It is interesting to note, therefore, that the first twelve cases seen in this institution were five years old or younger and in the remaining 34 cases there were 15 patients who were 14 years old or older and 5 of these succumbed. It is apparent from this survey that an increasing number of adults are being afflicted and that in many it is fatal. The age range in these cases is 2 months to 60 years. All of these were of the white race except two (nos. 16 and 39).

Symptomatology and Signs

The protean nature of this disease precludes the enumeration of signs and symptoms which are pathognomonic. Despite this, as will be noted in the table, most of the cases (55% in this series) are preceded by some type of upper respiratory infection which may antedate the onset of meningeal irritation by 1 day or 2 weeks. The actual onset of the meningitis is precipitous in most instances, manifested by headache, vomiting and ofttimes by convulsions or stupor. The signs depend upon the extent of meningeal involvement but the one constant finding as in all cases of meningitis, is some degree of nuchal rigidity. All this may sound very elementary but in hospital experience it is surprising to find that this particular sign sometimes is recognized by the physician two or three days before admission to an institution. Since early and prompt establishment of proper therapy is a prerequisite to a favorable prognosis, it is imperative that the above be kept in mind.

Diagnosis

The diagnosis hinges upon the identification of the H. influenza bacillus in the spinal fluid. This makes of the spinal tap an implement of diagnostic import which should be wielded not carelessly but promptly when nuchal rigidity is first suspected. The fluid should be examined both by smear and culture. The sugar content of the spinal fluid is usually decreased but may be normal, the protein is generally elevated and the cell count is increased but variable (in this group of cases 88 to 65,000) and polys usually pre-

dominate. Many times the blood culture is positive for B influenza but it is regrettable that only 14 cases in the present series had a culture of the blood performed. The prognostic value of this test must not be overlooked; of the five that were positive, there were only two who recovered.

There were five autopsies in this present group and the universal finding was a diffuse purulent meningitis.

Prognosis

According to Smith, et al,[2] Rivers reviewed the literature in 1922 and found a 92% mortality in 200 cases. With the advent of sulfapyridine and serum the recovery rate began to improve as shown by Neal[6] in 1940 in a list of 29 cases with 14 recoveries. Knouf et al[9] in a 10 year survey reported in 1942 an over-all mortality of 84% but in 12 cases treated with sulfapyridine and serum they showed a reduction to 25%. Subsequent reports show an excellent recovery rate of 80 to 100% with sulfanomides, serum, penicillin and streptomycin.[3,7,8,10–16]

The over-all mortality rate in this series was 28%. Eliminating those cases that were in the hospital less than 36 hours reduces the mortality rate to 21%. It is believed that this could be further decreased if the treatment outlined later in this discourse were to be followed.

The only sequelae noted were a hemiparesis in case no. 14, facial paralysis in no. 32 and vestibular nerve involvement in case no. 41.

As so clearly shown by Keefer[5] the unfavorable factors which portend a poor prognosis are bacteremia, extreme youth, delay in treatment, repeated convulsive seizures and associated brain abscess. This is substantiated by other investigators.

Treatment

Previous to the discovery of the sulfonamides treatment of influenzal meningitis was purely symptomatic. Sulfapyridine was the first effective sulfonamide. Fothergill's serum was widely used in the thirties but the results were discouraging. Following the introduction of type specific rabbit serum the mortality rates began to decrease, especially when combined with sulfadiazine. With the addition of penicillin and especially streptomycin to our armamentarium, treatment is now very satisfactory.

The dosage of type specific serum as outlined by Alexander[17] is dependent upon the sugar content of the spinal fluid. If the sugar is below 15 mg., the dosage of intravenous serum is 100 mg.; if over 40, it is 25 mg. Daily testing for capsular swelling of the organism is performed; if the serum of the patient in a 1:10 dilution does not produce swelling, more rabbit serum is given. Serum is sometimes effective when other measures fail (cases 8, 25, and 19) and should be used early in all cases in the author's opinion.

Sulfadiazine should be given intravenously at first to establish a prompt, satisfactory blood level, the dose being about 0.1 Gm/Kg. This drug is then given by mouth for two weeks after the temperature is normal

and spinal fluid negative, only in reduced dosage. This continued therapy is important because it is felt that some failures may be attributed to the early discontinuance of treatment (cases 4 and 27).

Penicillin has been employed in the usual dosages intraspinally and intramuscularly with good effect in some clinics, especially in England.[11-13] Unquestionably this medication is useful but its efficacy is limited. It should be used especially in those cases where the infecting organism proves to be sensitive and where it is desirous to prevent or treat secondary infection.

Streptomycin will effect many cures if given intrathecally and intramuscularly (50–100 mg. intraspinally and 1–2 Gm. in divided doses parenterally).[2,7,12,14–16] This antibiotic has been used in all cases in this series since it has become commercially available. Hoyne and Brown[7] believe that streptomycin intramuscularly and sulfonamides by mouth are all that are needed in the treatment of this disease. But Keefer[19] sounds a word of warning in the use of streptomycin. He finds that there may be serious complicating infections due to organisms resistant to streptomycin and suggests frequent bacteriological examinations of the nose and throat for same.

One means of therapy which is sometimes overlooked is whole blood transfusions. Not only does whole blood help to maintain the normal hematological elements but it also supplies a certain amount of antibodies which are effective against the influenzal bacillus.

Supportive care should be given as indicated such as oxygen, parenteral fluids, sedation, etc.

As a result of personal experience in the handling of many of the cases presented and in reviewing the remainder, the author would like to recommend the following procedures as outlined by Goodfriend and Thurston.[20] If the spinal fluid is cloudy at the time of the initial puncture, streptomycin and penicillin should be given intrathecally and both antibiotics started intramuscularly. At the same time sulfonamides intravenously and orally should be administered. Attempts to identify the organism are instituted immediately and sensitivity tests of the bacteria against the various antibiotics are determined. This combined treatment is continued until tests are completed and then the ineffective therapeutic agents are discontinued. In the opinion of the writer, type specific rabbit serum, too, should be used as soon as the organism is identified.

Summary

Forty-six cases of influenzal meningitis are presented from the services of a small community hospital. Considered as a rare cause of meningitis twenty years ago, this particular bacterium is now the leading organism invading the meninges. H. influenza, type B is the most frequent offender but two of the cases herein listed were due to type A.

Whereas this infection was once thought to be chiefly a disease of youth, it appears that an increasing number of adults are now being afflicted

with it and the mortality rate in this group is rising. The general recovery rate, however, is much better in recent years due to the combined use of the newer antibiotics and serum; a plan of treatment utilizing these products is offered.

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Steering Wheel Injuries of The Heart

Arnold H. Kadish,* M.D., Beverly Hills, Calif.

There are over fifty-three million drivers of automobiles† in the United States. Every driver is a potential victim of steering wheel injury. The purpose of this report is to call attention to trauma of the heart as a result of such injury and to point out that the expected frequency of contusion of the heart from this cause may be much greater than a review of the literature would indicate.

Review of the Literature

The literature contains many cases of direct nonpenetrating injuries to the heart, but in only a few is steering wheel trauma reported as the cause. Of those reported, the diagnosis was often pathologic rather than clinical. Reported cases of steering wheel injury to the heart are briefly tabulated on opposite page.

Perusal of the reported histories discloses several significant points. The most common early observation was tachycardia. This was noted in Beck's case in 2 of Elkin's patients and in 1 of Sigler's.

It would seem that the distress may be delayed, and that relatively long intervals may elapse before the development of cardiac signs—it is during this interval that treatment is imperative. For example, in Tuohy's case, the patient apparently was normal after the injury but died two weeks later while laughing heartily in a theatre. In Beck's case the patient persisted in minimizing the seriousness of his injuries; death occurrred on the the fifth day.

An interesting observation is that insufficient attention seems to have paid to nonpenetrating cardiac lesions which are less severe. From a review of the reported cases, however, it may be inferred that these may, nevertheless, be significant clinically. Certainly there is no reason to believe that the heart, situated as it is, is not subject to contusions of considerable variety. In Elkin's first case, the patient was able to leave the hospital in twenty-four hours, in his second case, the patient was apparently normal within three weeks, but his third patient, although physical examination revealed no abnormalities, complained of shortness of breath on exertion more than a year after the accident; unfortunately the further course of this third patient was not reported.

Pneumothorax may or may not be associated with cardiac injury from steering wheel trauma. Progressively increasing dyspnea or cyanosis point to pneumothorax, but the association of a cardiac injury should not be overlooked. Pneumothorax is promptly relieved by aspiration of air from the pleural cavity, and the patient may feel much better, but cardiac injury

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†Richardson, Lee C.: Bulletin American Association of Motor Vehicle Administrators, June, 1948.

*Instructor in Medicine, College of Medical Evangelists, Los Angeles, California.

REPORTED CASES OF STEERING WHEEL INJURY TO THE HEART *

Author	*Sex and Age of Patient*	*Clinical Picture*	*Results*	*Pathologic Examination*
Tuohy & Berdez[1] 1926	Man 63	Two weeks after injury: distress in chest; sudden collapse	Died 2 weeks after injury	Rupture in wall of left ventricle; pericardial cavity full of blood
Beck[2] 1935	Man 49	Pulse 136; patient persisted in minimizing seriousness of injuries; tinge of cyanosis; slight dyspnea; extrasystoles; inversion of T_1 and T_2	Died 5th day after injury	Contusions of posterior wall of left ventricle; laceration of myocardium
Elkin[3] (1)	Man 27	Pulse 134; slight cyanosis; bilateral pneumothorax without fracture of ribs; auricular fibrillation	Able to leave hospital in 24 hours; no symptoms since, even on strenuous exertion	
(2)	Man 18	Pulse 140; intense precordial pain requiring morphine; transient aortic insufficiency; blood pressure 140/30; diastolic aortic murmur; Corrigan pulse; electrocardiogram normal on third and tenth days	At end of 3 weeks had no abnormal symptoms or physical findings; no symptoms since	
(3)	Man 35	Onset with shock; pain in left side of chest with associated "tick-tick" cardiac sounds in which 1st and 2nd sounds could not be differentiated	Examination of heart normal in 3 weeks, but patient complained of shortness of breath on exertion 1 year after injury	
Sigler[4] (1) 1942	Man 21	Pulse 120 and poor in quality; precordial pain; progressive increase in cardiac size; associated dyspnea; pericardial friction rub 14 days after admission	Complete recovery within 4 months	
Sigler[5] (2)	Man 46	Little shock; pain in sternal region on breathing or moving around; in hospital 10 days; electrocardiogram 6 days after accident had definitely lowered T waves in all leads as compared with previous normal electrocardiograms	Electrocardiogram returned to normal in 7 weeks; clinically, feeling fine	

must be sought. This association was reported in Elkin's first case and, it will be noted, without fracture of the ribs.

The development of transient murmurs following injury, particularly aortic insufficiency with its peripheral signs (Corrigan pulse, etc.) should bring to mind the possibility of traumatic valvular rupture. Such was the case in Elkin's second patient. Similar cases have come to the autopsy table.[6]

REPORT OF A CASE

Mr. E., 53 years of age, who had always been in good health, an enthusiastic horseback rider, who "used to break horses in" until very recently, had an automobile accident on the evening of April 28, 1948. A woman coming from the opposite direction lost control of her car during a rain storm and crossed the road in front of him. Mr. E., who was driving slowly, was thrown forward against the steering wheel as his car hit the other broadside. Later, it was learned that the steering wheel was bent and had to be replaced. Although somewhat dazed, he did not feel ill. He was greatly concerned about his wife, who have been cut by the windshield and who had received emergency first aid at the time of the accident; he brought her to my office for medical care the next day. At first he declined to be examined himself but finally was persuaded by his wife to consent.

General examination did not discover anything of significance save a tachycardia; his pulse was 110. An electrocardiogram, however, disclosed inversion of the T waves in all precordial leads as well as a low T_1. Because of the nature of the injury, a roentgenogram of the chest was ordered but proved to be essentially normal. Two days after the accident, on April 30, he suffered several attacks of substernal pain, moderate in severity, oppressive in character and radiating down the inner aspects of both arms so that they felt, to quote him, "as though they were weighted down with lead." These attacks lasted only a few minutes and were not related to exertion. The third day following the injury, May 1, he was seized by a severe substernal pain which lasted twenty minutes and radiated into both arms as before. He felt faint, belched frequently, appeared pale and perspired freely. There were, however, no further electrocardiographic changes. On May 10 a fluoroscopic examination of the heart disclosed a localized area of paradoxical pulsation about 2 cm. in diameter, the amplitude of which was 3 to 4 mm., on the anterior lateral aspect of the left ventricular border.

The patient was placed on a program of complete mental and physical rest for three weeks with a period of gradually increasing activity following this. An electrocardiogram made approximately three and a half months later (August 19) was normal.

SUMMARY AND CONCLUSIONS

The expected frequency of cardiac injury subsequent to steering wheel trauma may be much greater than a review of the literature would indicate.

It would seem that many more cases may occur than are diagnosed clinically.

Distress and the development of cardiac signs may be delayed for a relatively long interval following steering wheel injury.

Steering wheel trauma to the heart should be suspected and sought in every driver of an automobile who is involved in an accident. Tachycardia, particularly, should be noted. Electrocardiographic studies including the precordial leads should be carried out. Fluoroscopic studies may be of diagnostic aid in certain cases.

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News, Notes and Editorial Comments

Muracil Is New Name for Antibason—Now Council-accepted

Organon has changed the name of its brand of methyl thiouracil from Antibason to MURACIL. This change has been made to conform with the nomenclature of the Council of Pharmacy and Chemistry of the American Medical Association which body has just accepted MURACIL as the first brand of methyl thiouracil for inclusion in New and Nonofficial Remedies.

Physicians should bear this new name—MURACIL—in mind when prescribing this Council-accepted brand of methyl thiouracil for their patients with hyperthyroidism. MURACIL (Formerly Antibason) will continue to be available for physicians' prescriptions in 50-mg. tablets, bottles of 1000 and 100 tablets.

New Method of Diagnosing Polio Is Sought

A March of Dimes grant of $17,200 will enable scientists at New York University-Bellevue Medical Center to continue their attempts to devise a method of diagnosis for polio. This was announced jointly today by Basil O'Connor, President of the National Foundation for Infantile Paralysis and Dr. Donal Sheehan, Chairman of the Center's Scientific Committee. Heading the research project, which was begun in 1945 with National Foundation support, will be Dr. Robert Ward, Professor of Pediatrics at New York University.

Dr. Ward will concentrate his efforts on developing a test-tube method for the diagnosis of polio, using complement fixation methods similar to those routinely used to diagnose other virus diseases. The New York scientist's aim is to purify and concentrate the virus used in the test. Because the only certain methods of identifying polio at the present time require the use of many laboratory animals and take weeks to complete, Dr. Ward is trying to develop a rapid, simple test. This has long been the goal of many scientists.

Twenty-second Anniversary Number of Harofe Haivri
The Hebrew Medical Journal

The appearance of Volume 2, 1949, of *The Hebrew Medical Journal* (Harofe Haivri), concludes the 22nd year of publication of this bi-lingual, semi-annual Journal, edited by Moses Einhorn, M.D. Written in Hebrew, with English summaries, the Journal is a contribution to the development of Hebrew medical literature and thus facilitates teaching in the newly established Hebrew University-Haddassah Medical School in Israel. In this issue a detailed article is presented on "Scoliosis" by Samuel Kleinberg, M. D., and Prof. Arnold Kutzinski gives a comprehensive survey on "The Psychopathological Problems of the Jews in Israel."

There is a special section devoted to Historical Medicine which contains three interesting essays: "Medical and Anatomical Terms in the Pentateuch in the Light of Egyptian Medical Papyri" by Prof. A. S. Yahuda: "Jews as Intermediaries of Medicine and Natural Science During the Middle Ages" by Zussmann Munter, M.D.; "Al-Qirqisani's Essay on the Psycho-Physiology of Sleep and Dreams" by Dr. Leon Nemoy. Under the heading of "Personalia" there are several articles paying tribute to Dr. Solomon R. Kagan on the occasion of his sixtieth birthday. Dr. Kagan is a well-known authority on Jewish medicine, medical biography and bibliography. For further information, communicate with the Editorial Office of *The Hebrew Medical Journal*, 983 Park Avenue, New York 28, N. Y.

The release of new laboratory reagents for use in preparation of Rh blood grouping sera is announced by Sharp & Dohme, Inc., Philadelphia. Specific Polysaccharides Blood Group A and Blood Group B (Reagents) are concentrated, sterile solutions of complex polysaccharides that neutralize the anti-A and anti-B agglutinins in Rh blood grouping sera.

These new reagents perform this action without affecting the stability of anti-Rh sera or the activity of the anti-Rh agglutinin. They also contain a preservative that has no adverse effect on Rh agglutinins. The desired amount of either reagent is withdrawn in a sterile syringe and injected into the Rh serum being prepared. The mixture is then shaken thoroughly to obtain a uniform preparation. Specific Polysaccharides Blood Group A and Blood Group B (Reagents) are supplied separately in packages of six 1-cc. vials and one 10-cc. vial.

General Armstrong Sworn in as Air Force Surgeon General

Major General Harry G. Armstrong was sworn in on December 9, 1949 as Surgeon General of the U. S. Air Force Medical Service to succeed Major General Malcolm C. Grow, who retired on November 30. The Air Judge Advocate General, Major General Reginald C. Harmon administered the oath in the office of USAF Chief of Staff, General Hoyt S. Vanderberg.

General Armstrong, an authority on aviation medicine, was a winner of the Collier Award for aviation achievement in 1939. He first took an interest in the relation of medicine to flying while in private practice in Minneapolis, Minnesota, after his graduation from the University of Louisville Medical School in 1925.

Both General Armstrong and General Grow were assigned to the Engineering Division of the Air Corps at Wright Field, Dayton, Ohio, in 1934, when they became active in establishing an aviation medicine research laboratory that grew into the present-day Aero Medical Laboratory there. General Armstrong was director of the Aero Medical Laboratory for six years, and was responsible for its rapid growth and many accomplishments.

General Armstrong initiated research into the effects of flying on the body while director of the laboratory. He made the first detailed study of the effect of noise, and changes in altitude on the ear, and pioneered in demonstrating the effects of lack of oxygen at progressively higher altitudes. He adapted a mask for aircrews designed by Doctors Ed Boothby and W. Randolph Lovelace, II, of the Mayo Clinic; and initiated development of the oxygen system now in use in aircraft.

General Armstrong's recommendations for improved aircraft design and making flight at high altitudes safer and reducing pilot fatigue, for the use of protective clothing and equipment, were the basis for his being awarded the Collier Award in 1939. He prepared the medical specifications and did the test work on the first successful pressure cabin airplane, and personally participated in experiments to determine what happens when the cabin is suddenly decompressed at high altitude.

It was also in 1939 that General Armstrong published "Principles and Practices of Aviation Medicine," the first inclusive text on that subject. The book is not only a standard work for flight surgeons, but has been highly beneficial to airplane designers, pilots, and medical students.

While a Reserve officer in the Army Medical Corps in 1929, he entered the Air Force School of Aviation Medicine, then at Brooks Field, Texas, as a student. It was there that he made the first free-fall parachute jump in medical records expressly to discover the sensations of a free fall. On this occasion he jumped at a height of 10,000 feet, but did not pull his rip cord to open the chute until he was at 3,000 feet.

During the war, General Armstrong served as Surgeon of the Eighth Air Force, based in England. Here he instituted the use of rest homes for

combat crews, improved the protective clothing in use, and put into operation the first Central Medical Establishment. Here much of the research on frost-bite at high altitudes, survival and air-rescue problems, and death from loss of oxygen supply was carried out.

After the war, General Armstrong, while with the Office of Military Government in Berlin, visited every European country and studied advances made in medicine during the war years. He was one of the group which established an aeromedical laboratory in Heidelberg. In 1946 he became Commandant of the School of Aviation Medicine at Randolph Air Force Base, Texas, where, in 1941, he had established the Research Section. As Commandant, General Armstrong began research in the newest medical field when he organized the Department of Space Medicine. This was done in anticipation of possible engineering developments which might make it practical to design manned rocket ships capable of flight beyond the earth's atmosphere. In July, 1949, General Armstrong became Deputy Surgeon General.

He has received numerous degrees, including the Master of Arts in Internal Medicine from the University of Cincinnati; Master of Science in Physiology from the University of Toronto, Toronto, Canada; and has twice been given an honorary Doctorate of Science, once by the University of South Dakota and once by the University of Louisville. He has been awarded the Legion of Merit, with oak leaf cluster, the Cruses de Guerre, with palms, of Belgium and France, and the Order of the British Empire. General Armstrong is a native of De Smet, South Dakota. He is married to the former Mary E. Sutherland, of Harrodsburg, Kentucky, and has two children, a daughter, Jeff, aged 21, and a son, Bradley, aged 19.

BOOK REVIEWS

MANUAL OF MEDICAL EMERGENCIES, by Stuart C. Cullen, M.D., and E. G. Gross, M.D., published by the Year Book Publishers, Inc., 1949.

This manual is designed to cover only the acute emergency; extensive diagnostic measures and continued definitive therapeutic measures are deliberately excluded. The material is presented in the belief that many patients' lives can be saved with appropriate emergency therapeutic drugs and procedures efficiently applied until complete diagnosis is possible and specific therapy is determined. An invaluable guide in everyday practice.

THE DYNAMICS OF PARENT-CHILD RELATIONSHIPS, by Percival M. Symonds, published by Teachers College, Columbia University, 1949.

This book is intended for use by counselors and psychotherapists who work in child guidance and in parent education and psychotherapy. It may appropriately be used as a supplement to and as collateral reading with the author's *The Dynamics of Human Adjustment.*

The studies that have led up to this book indicate that the development of the child's personality is closely related to his relationships with his parents. This is a conclusion of the utmost importance. It means that one must consider parent-child relationships not only with respect to their influence in causing mental and emotional disorders and psychopathic states but also in connection with the development of normality, leadership and genius. It means that attention must be given increasingly to the problems of parent-child relationships even to their most minute details, for it is in the small and subtle aspects of these relationships that the security of the child develops.

The essence of parent-child relations, it must be emphasized lies more in how a parent feels than in what a parent does. If a parent truly loves his child, considerable departure from recommended practices in the handling of the child will not lead to unfavorable personality development. Helping to influence parents in what they do is the province of parent education and has long been understood, helping to influence parents in how they feel, less well understood, is the province of parent psychotherapy. Both parent education and parent psychotherapy are important: but for the cultivation of constructive parent-child relationships and the development of desirable child personality, influencing parent feeling is more important than influencing parent behavior.

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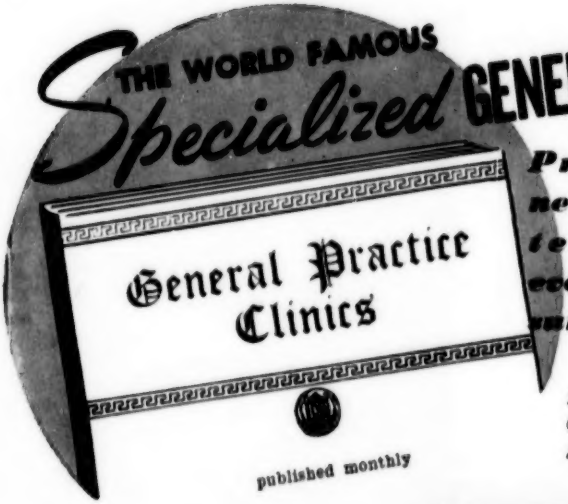

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